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PubMed☐ 1: Caries Res 1997;31(4):268-74Related Articles, [Books](#), [LinkOut](#)PubMed
Services**Passive immunization against dental plaque formation in humans: effect of a mouth rinse containing egg yolk antibodies (IgY) specific to Streptococcus mutans.****Hatta H, Tsuda K, Ozeki M, Kim M, Yamamoto T, Otake S, Hirasawa M, Katz J, Childers NK, Michalek SM.**

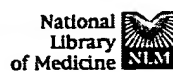
Taiyo Kagaku Co., Ltd., Central Research Laboratories, Mie, Japan.

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Passive immunization involving the delivery of antibodies specific to pathogens of infectious diseases to the host has been an attractive approach to establish protective immunity against a variety of microbial pathogens, including Streptococcus mutans, which is the principal etiologic agent of dental caries in humans. The overall purpose of the present study was to determine the effectiveness of a mouth rinse containing antibodies to S. mutans in preventing the establishment of this bacterium in dental plaque of humans. The antibodies were derived from egg yolks obtained from hens immunized with whole cells of S. mutans grown in sucrose-containing medium. The immunoglobulin derived from the yolks (IgY) of immunized hens was characterized in vitro and in vivo in human volunteers. Cross-reactivity tests showed that immune IgY reacted with every serotype, except serotype b, which had lost its GTase activity, when the bacteria were cultured in sucrose-containing medium. Immune IgY inhibited S. mutans adherence to saliva-coated hydroxyapatite discs by 59.2%, while control IgY caused an inhibition of only 8.2%. In the short-term (4-hour) test using a mouth rinse containing 10% sucrose, immune IgY decreased the ratio of the percentage of S. mutans per total streptococci in saliva. In the long-term (7-day) test using a mouth rinse without sucrose, the ratio in saliva was not significantly reduced in the volunteers using the immune IgY due to the large standard deviation. However, comparing the ratios of the percentage of S. mutans per total streptococci in plaque of individual subjects, there was a tendency for a reduction of the ratios in the volunteers receiving the mouth rinse containing immune IgY. These results support the effectiveness of IgY with specificity to S. mutans grown in the presence of sucrose as an efficient method to control the colonization of mutans streptococci in the oral cavity of humans.

Publication Types:

- Clinical Trial
- Controlled Clinical Trial



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PubMed☐ 1: Infect Immun 1991 Nov;59(11):4161-7Related Articles, [Books](#), [LinkOut](#)PubMed
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Oral passive immunization against dental caries in rats by use of hen egg yolk antibodies specific for cell-associated glucosyltransferase of *Streptococcus mutans*.

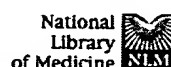
Hamada S, Horikoshi T, Minami T, Kawabata S, Hiraoka J, Fujiwara T, Ooshima T.

Department of Oral Microbiology, Osaka University Faculty of Dentistry, Japan.

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The effect of polyclonal egg yolk immunoglobulin G antibodies (yIgG) raised against whole cells, cell-free (CF) glucosyltransferase (GTase), or cell-associated (CA) GTase of serotype c *Streptococcus mutans* was examined in terms of in vitro inhibition of virulence-related factors of *S. mutans* and protection of *S. mutans*-infected rats against the development of dental caries. Hens (18 weeks old) were immunized with formalin-treated whole cells, purified CF-GTase, or CA-GTase together with Freund's complete adjuvant. In addition, yIgG to surface protein antigen was used in some in vitro experiments for comparison with other antibodies. yIgG was purified by ammonium sulfate precipitation followed by DEAE-Sephacel column chromatography or fractional precipitation with ethanol. Purified yIgG was found to be a 220-kDa protein, which was dissociated into heavy and light chains upon addition of 2-mercaptoethanol. yIgG to whole cells and surface protein antigen gave a heavy aggregation of *S. mutans* organisms, while yIgG to CF- and CA-GTase specifically inhibited the enzymatic activity of the respective GTase. yIgG to CA-GTase and whole cells was found to clearly suppress the adherence of *S. mutans* cells to a glass surface. Specific-pathogen-free Sprague-Dawley rats that had been infected heavily and repeatedly with *S. mutans* and fed diet no. 2000 developed severe dental caries, while rats fed diet 2000 containing greater than or equal to 0.1% yIgG to CA-GTase showed a statistically significant reduction in dental plaque accumulation and caries development. Administration of yIgG to CF-GTase and whole cells failed to protect against caries. These results clearly suggest that yIgG to *S. mutans* CA-GTase specifically inhibited a virulence factor of this organism, i.e., insoluble glucan-synthesizing CA-GTase, resulting in a significant reduction in the development of dental caries.

PMID: 1834573 [PubMed - indexed for MEDLINE]



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PubMed☐ 1: J Dent Res 1991 Mar;70(3):162-6Related Articles, ~~Books~~ Books, LinkOut

Protection of rats against dental caries by passive immunization with hen-egg-yolk antibody (IgY).

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Otake S, Nishihara Y, Makimura M, Hatta H, Kim M, Yamamoto T, Hirasawa M.

Department of Clinical Pathology, Nihon University School of Dentistry, Chiba, Japan.

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Hen-egg-yolk antibody (IgY) was prepared against *Streptococcus mutans* MT8148 serotype c that was cultivated in medium containing sucrose, and it was used in passive caries-immunity studies. Specific pathogen-free rats infected with *S. mutans* MT8148 (c) and fed with a cariogenic diet containing more than 2% immune yolk powder developed significantly lower caries scores than did the ones infected with the same strain and fed with a diet containing only control yolk powder obtained from non-immunized hens. Similar results were obtained in an experiment with rats infected with *S. mutans* JC-2 (c) strain. Rats provided a diet supplemented with 0.5% immune water-soluble protein fraction containing *S. mutans*-specific IgY and challenged with *S. mutans* MT8148 exhibited significantly fewer caries lesions, compared with control rats on the normal diet.

PMID: 1825668 [PubMed - indexed for MEDLINE]

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☐ 1: Oral Microbiol Immunol 1999 Feb;14(1):1-20 Related Articles, [Books](#), LinkOut

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Current status of a mucosal vaccine against dental caries.

Hajishengallis G, Michalek SM.

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Department of Oral Biology, University of Alabama, Birmingham 35294-2170, USA.

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The evidence of a specific bacterial cause of dental caries and of the function of the salivary glands as an effector site of the mucosal immune system has provided a scientific basis for the development of a vaccine against this highly prevalent and costly oral disease. Research efforts towards developing an effective and safe caries vaccine have been facilitated by progress in molecular biology, with the cloning and functional characterization of virulence factors from mutans streptococci, the principal causative agent of dental caries, and advancements in mucosal immunology, including the development of sophisticated antigen delivery systems and adjuvants that stimulate the induction of salivary immunoglobulin A antibody responses. Cell-surface fibrillar proteins, which mediate adherence to the salivary pellicle, and glucosyltransferase enzymes, which synthesize adhesive glucans and allow microbial accumulation, are virulence components of mutans streptococci, and primary candidates for a human caries vaccine. Infants, representing the primary target population for a caries vaccine, become mucosally immunocompetent and secrete salivary immunoglobulin A antibodies during the first weeks after birth, whereas mutans streptococci colonize the tooth surfaces at a discrete time period that extends around 26 months of life. Therefore, immunization when infants are about one year old may establish effective immunity against an ensuing colonization attempts by mutans streptococci. The present review critically evaluates recent progress in this field of dental research and attempts to stress the protective potential as well as limitations of caries immunization.

Publication Types:

- Review
- Review, Tutorial

PMID: 10204475 [PubMed - indexed for MEDLINE]

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PubMed ID: 1834573 Feb 12, 2002 10:33:18

PMID: 9197932 [PubMed - indexed for MEDLINE]

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